

Induction of endoplasmic reticulum stress pathway by green tea epigallocatechin-3-gallate (EGCG) in colorectal cancer cells: activation of PERK/p-eIF2 α /ATF4 and IRE1 α

ABSTRACT

Epigallocatechin-3-gallate (EGCG) is the most abundant bioactive polyphenolic compound among the green tea constituents and has been identified as a potential anticancer agent in colorectal cancer (CRC) studies. This study was aimed to determine the mechanism of actions of EGCG when targeting the endoplasmic reticulum (ER) stress pathway in CRC. The MTT (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyl tetrazolium bromide) assay was performed on HT-29 cell line and normal cell line (3T3) to determine the EGCG toxicity. Next, western blot was done to observe the expression of the related proteins for the ER stress pathway. The Caspase 3/7 assay was performed to determine the apoptosis induced by EGCG. The results demonstrated that EGCG treatment was toxic to the HT-29 cell line. EGCG induced ER stress in HT-29 by upregulating immunoglobulin-binding (BiP), PKR-like endoplasmic reticulum kinase (PERK), phosphorylation of eukaryotic initiation factor 2 α subunit (eIF2 α), activating transcription 4 (ATF4), and inositol-requiring kinase 1 α (IRE1 α). Apoptosis was induced in HT-29 cells after the EGCG treatment, as shown by the Caspase 3/7 activity. This study indicates that green tea EGCG has the potential to inhibit colorectal cancer cells through the induction of ER stress.